ASSESSMENT REPORT ON

ACRYLONITRILE

FOR DEVELOPING AN AMBIENT AIR QUALITY GUIDELINE

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FOREWORD

Alberta Environment maintains Ambient Air Quality Guidelines to support air quality management in Alberta. Alberta Environment currently has ambient guidelines for thirty-one substances and five related parameters. These guidelines are periodically updated and new guidelines are developed as required. Fact Sheets on Ambient Air Quality Guidelines were updated in September 1997 and February 2000.

With the assistance of the Clean Air Strategic Alliance, a multi-stakeholder workshop was held in October 2000 to set Alberta's priorities for the next three years. Based on those recommendations, a three-year work plan ending March 31, 2004 was developed to review four existing guidelines, create three new guidelines for three families of substances, and adopt six new guidelines from other jurisdictions.

This document is one in a series of documents that presents the scientific assessment for these substances.

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SUMMARY

Acrylonitrile is a volatile and flammable gas. It does not occur naturally and there is no evidence that chemical reactions may lead to its formation in the atmosphere. It is used primarily in the fabrication of acrylic fiber, resins, rubbers, and elastomers. The major industrial sectors contributing to acrylonitrile emissions in Canada, in order of importance, are the chemical and chemical products sector, transportation equipment industries and plastic product industries. The National Pollutant Release Inventory reports that there are no major emissions of acrylonitrile from industrial facilities in Alberta.

No published literature could be found on the direct effects (through the atmosphere) of acrylonitrile exposure to terrestrial plants. The primary route of acrylonitrile exposure for the general public and animals would be through inhalation. However, it is unlikely that significant exposure to acrylonitrile would occur in the absence of an industrial source or hazardous waste facility emitting this substance.

Adverse health responses have been reported in humans after acute (accidental) exposure via inhalation and/or skin contact. These responses include eye and throat irritation, central nervous system effects (e.g. headaches, nausea, vomiting, convulsions and unconsciousness), and death. Corresponding doses (exposure concentrations and durations) associated with these events are not known with a great deal of certainty since exposures were accidental and air concentrations could not be measured. With regard to acute, short-term exposure, animal studies indicate that acrylonitrile has caused salivation, tearing of the eyes, diarrhea, gastrointestinal injury, seizures, and death. These types of responses have been observed at concentrations ranging from 58 to 1,330 mg/m^3 (26 to 600 ppm) over exposure durations ranging from 4 hours to 60 hours (on an intermittent basis).

Chronic, long-term exposures have been demonstrated to cause different cancers in a number of rat studies, including tumour formation in the brain and spinal cord, small intestine, tongue, Zymbal gland and mammary glands. Air concentrations associated with these cancers ranged from 45 to 178 mg/m^3 (20 to 80 ppm) for exposure durations of one to two years (on an intermittent basis). Long-term human exposure has occurred in occupational settings. Results of these studies are inconclusive in terms of consistently establishing acrylonitrile's ability to cause cancer because of confounding factors (specifically, exposure to other chemicals and lifestyle factors such as smoking). Notwithstanding, data from these studies are used by US Environmental Protection Agency to represent excess incidence of respiratory cancers in humans corresponding to 33 mg/m^3 (15 ppm) as an 8-hour time weighted average concentration for an average exposure duration of 9 years. Most regulatory agencies consider acrylonitrile to be potentially carcinogenic to humans due to evidence of carcinogenicity in rat studies.

Chronic, long-term exposures have been demonstrated to cause different adverse responses in several rat studies, including irritation of eye and skin; developmental, neurological, respiratory, and systemic effects; and death. Air concentrations associated with these responses ranged from 45 to 178 mg/m^3 (20 to 80 ppm) for exposure durations of one to two years (on an intermittent basis).

The majority of agencies reviewed as part of this assessment do not have an air quality guideline for acrylonitrile for acute, short-term (<24 hours) exposure conditions. There are exceptions, as several US agencies have 24-hour guidelines ranging from 2 to 217 $\mu g/m^3$ (0.9 to 100 ppb) for specific purposes. These include the US Agency for Toxic Substances and Disease Registry and state agencies in Michigan, New Hampshire, and Oklahoma. The approaches used by these agencies in developing their guidelines involved non-carcinogenic risk assessment procedures or taking an occupational exposure level and dividing it by safety or adjustment factors. The province of Ontario uses a maximum point of impingement guideline of 180 $\mu g/m^3$ (83 ppb) for a 30-min. averaging time.

All agencies other than the US Agency for Toxic Substances and Disease Registry have chronic, long-term (>1 year) guidelines for acrylonitrile. Almost all of these agencies use cancer as the basis to derive their respective guideline – based upon the US Environmental Protection Agency's inhalation unit cancer risk factor of 6.8E-05 per $\mu g/m^3$. Using this unit risk factor and an increased lifetime cancer risk of 1 in 100,000 (a risk criterion commonly used in Alberta), an air concentration of 0.15 $\mu g/m^3$ is estimated for acrylonitrile. This corresponds to the guideline level for cancer used by most of these agencies. Some of the agencies use a non-cancer endpoint developed by the US Environmental Protection Agency (reference concentration of 2 $\mu g/m^3$ or 0.9 ppb) as a chronic air guideline.

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1.0 INTRODUCTION

Alberta Environment (AENV) establishes Ambient Air Quality Guidelines under Section 14 of the Environmental Protection and Enhancement Act (EPEA). These guidelines are part of the Alberta air quality management system (AENV, 2000).

The main objective of this assessment report is to provide a review of scientific and technical information to assist in evaluating the basis and background for an Ambient Air Quality Guideline for acrylonitrile. The following aspects were examined as part of the review:

- physical and chemical properties,
- existing and potential natural and anthropogenic emissions sources in Alberta,
- effects on humans, animals, and vegetation,
- monitoring techniques,
- ambient air guidelines in other Canadian jurisdictions, United States, European Union and Australia, and the basis for development and use.

Important physical and chemical properties that govern the behaviour of acrylonitrile in the environment include, but are not limited to, chemical structure, molecular weight, melting and boiling points, water solubility, density, vapor density, organic carbon partition coefficient, octanol water partition coefficient, vapor pressure, Henry's Law constant, bioconcentration factor, and odor threshold. Values for these properties will be reviewed and presented in this report.

Existing and potential natural and anthropogenic sources of acrylonitrile emissions in Alberta will also be presented. Natural emissions of acrylonitrile are described in the literature. Anthropogenic emissions are provided in Environment Canada's National Pollutant Release Inventory (NPRI).

Scientific information about the effects of acrylonitrile on humans and animals is reported in published literature and other sources. This information includes toxicological studies published in professional journals and reviews and information available through the Agency for Toxic Substances and Disease Registry (ATSDR), Environment Canada, International Agency for Research on Cancer (IARC), and US Environmental Protection Agency's Integrated Risk Information System (IRIS). All of these sources provide valuable information for understanding health effects of acrylonitrile exposure.

Reference air monitoring and other techniques for detecting acrylonitrile in air are also documented in published literature. A number of widely used and accepted reference methods exist for acrylonitrile. These methods have been developed, tested and reported by US Environmental Protection Agency (US EPA), US National Institute of Occupational Safety and Health (NIOSH), and US Occupational Safety and Health Administration (OSHA).

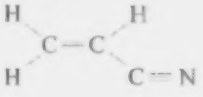
2.0 GENERAL SUBSTANCE INFORMATION

Acrylonitrile is a colourless to slightly yellow liquid at ambient temperature and pressure (IPCS, 1983). Its odour has been described as sweet and faintly pungent (IPCS, 1983), resembling that of peach seeds (SRC, 1985), onion and garlic (Verschueren, 2001; ATSDR, 1990) or mustard (FAO, 1965).

Acrylonitrile is volatile and flammable (IPCS, 1983). When heated, decomposition of acrylonitrile produces harmful gases, such as cyanide (EC, 2001; Lewis, 2000; IPCS, 1986) and NO_x (Lewis, 2000). Acrylonitrile may undergo a number of different reactions via its carbon-carbon double bond or cyano (- CN) group. These reactions include polymerization, hydration, cyanoethylation and oxidation (IPCS, 1983). In the absence of oxygen, spontaneous explosive polymerization of acrylonitrile may occur if exposed to light, alkali (IPCS, 1983) or silver nitrate (Lewis, 2000). Acrylonitrile reacts violently with strong oxidizers, strong acids, strong bases (Lewis, 2000; IPCS, 1986), azoisobutyronitrile, diobenzoyl peroxide, di-tert-butylperoxide or bromine (Lewis, 2000).

Table 2-1 provides a list of common synonyms, trade names and a list of important identification numbers for acrylonitrile.

Table 2-1 Identification of Acrylonitrile

Property	Value
Formula	C_3H_3N
Structure	
CAS Registry number	107-13-11
RTECS number	AT5250000
UN Number	UN1093
Common Synonyms	acrylic nitrile AN Cyanoethylene 2-propenenitrile VCN vinyl cyanide
Trade names	Acritet Acrylon Carbacryl Caswell No. 010 ENT 54 Fumigrain Miller's fumigrain TL 314 Ventox

In general, acrylonitrile is used primarily in the fabrication of acrylic fiber, acrylonitrile-butadiene-styrene (ABS) resins, adiponitrile, nitrile rubbers, elastomers and styrene-acrylonitrile (SAN) resins (IARC, 1999). Of these, the most important use of acrylonitrile is as the raw material in the fabrication of acrylic fibers for the clothing, and furnishings (IARC, 1999). Other novel applications for acrylonitrile include the manufacturing of carbon fibers and the production of fatty amines, ion exchange resins and fatty amine amides (IARC, 1999). In the past, acrylonitrile, in combination with carbon tetrachloride, was used as a fumigant and as a pesticide, but these applications have ceased (IARC, 1999; ATSDR, 1990).

Acrylonitrile is not produced in Canada but it is imported and used in Canada (EC, 2000). The majority of acrylonitrile imported into Canada is used in the production of nitrile-butadiene rubber while some is used in the production of ABS and SAN resins (EC, 2000). Only a small fraction of the acrylonitrile imported into Canada is used for other miscellaneous applications (EC, 2000). According to Environment Canada (2000), acrylonitrile is not used in Canada to produce polyacrylic fibers.

2.1 Physical, Chemical and Biological Properties

The physical and chemical properties of acrylonitrile are summarized in Table 2-2.

2.2 Environmental Fate

The environmental fate of acrylonitrile is summarized in Table 2-3. When acrylonitrile is released into the atmosphere, it undergoes a photochemical reaction with hydroxyl radicals. Reaction of acrylonitrile with hydroxyl radicals mainly produces formaldehyde but also other compounds, such as formic acid, formyl cyanide, carbon monoxide and hydrogen cyanide (EC, 2000). Since the half life is of the order of days, acrylonitrile may disperse from the source areas (Genium, 1999). Acrylonitrile released to water will slowly volatilize and may biodegrade (Genium, 1999). Due to its high Henry's Law constant and low adsorption to soil (EC, 2000; Genium, 1999), acrylonitrile will volatilize quickly when spilled on soil (Genium, 1999) or may leach into the groundwater (Ontario MOE, 2000). Bioaccumulation is considered negligible (EC, 2000; IPCS, 1983).

Table 2-2 Physical and Chemical Properties of Acrylonitrile

Property	Value	Reference
Molecular Weight	53.06	Lide, 2001
Physical state	Liquid	
Melting Point	-83.5 °C	Lide, 2001
	-82 °C	Howard, 1989
Boiling Point	77.3 °C	Lide, 2001; Lewis, 2000; Genium, 1999; Lewis, 1993; Howard, 1989
Specific gravity (liquid)	0.806 at 20 °C	Lide, 2001; Ontario MOE, 2001; ATSDR, 1990
	0.8004 at 25 °C	Genium, 1999; Lewis, 1993
Specific gravity (gas) (air =1)	1.9	Ontario MOE, 2001
	1.83	Verschueren, 2001; Lewis, 2000; Genium, 1999
Vapour pressure	100 mm Hg at 23 °C	Ontario MOE, 2001; Genium, 1999; Lewis, 2000; ATSDR, 1990
	137 mm Hg at 30 °C	Verschueren, 2001
	11,000 to 15,600 Pa	Mackay et al., 1995
	107.8 mm Hg at 25 °C	Howard, 1989
Solubility in water	73,500 mg/L	Ontario MOE, 2001
	73,240 to 216160 mg/L	Mackay et al. 1995
	79,000 mg/L	ATSDR, 1990
	75,000 mg/L	Howard, 1989
Solubility	Soluble in all common organic solvents	Lewis, 1993
Henry's Law Constant	$8.8x10^{-5}$ atm.m^3/mol	Ontario MOE, 2001; ATSDR, 1990
	0.063	Genium, 1999
	8.918 to 11.14 Pa.m^3/mol	EC, 2000; Mackay et al., 1995
	$1.10x10^{-4}$ atm.m^3/mol	Howard, 1989
	1.38x10-4 atm.m^3/mol	Bocek, 1976
Octanol water partition coefficient (log K_{ow})	-0.92	Verschueren, 2001; Lech et al., 1995; ATSDR, 1990;IPCS, 1983
	0.25	Genium, 1999; Howard, 1989
	-0.92 to 1.2	EC, 2000
	-0.92 to 0.30	Mackay et al., 1995
Organic carbon partition coefficient (Log K_{oc})	9	Genium, 1999; Howard, 1989
	-0.07	ATSDR, 1990
	-0.0899 to 1.101	Mackay et al., 1995
Flash Point (closed cup)	0 °C	Lide, 2001; Ontario MOE, 2001; IPCS, 1983; Lewis, 1993
	-1 °C	Genium, 1999; ATSDR, 1990
Explosive limits	3.0% to 17.0%	Lide, 2001; Genium, 1999; Lewis, 1993
Autoignition temperature	481 °C	Lide, 2001; Genium, 1999
Odour threshold	8.100 to 78.75 mg/m^3	Genium, 1999
Bioconcentration factor in fish (log BCF)	1 (estimated)	Howard, 1989
	48 (experimental)	Genium, 1999; Howard, 1989
	0.00 to 1.68	Mackay et al., 1995
	0.48 to 1.68	EC, 2000
Conversion factors for vapour (at 25 °C and 101.3 kPa)	1 ppm = 2.17 mg/m^3	Ontario MOE, 2001; IPCS, 1983; 1986
	1 ppm = 2.203 mg/m^3	Verschueren, 2001; ATSDR, 1990
	1 mg/m^3 = 0.4605 ppm	IPCS, 1983; 1986
	1 mg/m^3 = 0.454 ppm	Verschueren, 2001; ATSDR, 1990

Table 2-3 Environmental Fate of Acrylonitrile (EC, 2000; Genium, 1999; Mackay et al., 1995; Howard, 1989; IPCS, 1983)

System	Fate	Half life
Water	Slow biodegradation from one week to 20 days; loss by volatilization; biodegradation by acclimatized microorganisms; adsorption to sediment or suspended particulate matter and bioconcentration to aquatic organisms is negligible;	• *In water:* 5 to 7 days • *Due to volatilization*: 1.2 to 6 days • *Biodegradation:* complete biodegradation in approximately 1 week by acclimatized microorganisms, half life of 30 to 552 hours
Soil	Rapid volatilization; negligible adsorption to soil; potential for leaching into groundwater	
Air	Degradation by reaction with photochemically produced hydroxyl radicals	• *Reaction with hydroxyl radicals:* 3.5 days • *Photooxidation:* 4 hours (in the troposphere), 13.9-149 hours • *Atmospheric transformation*: 1-5 to more than 5 days, 9 to 10 hours

3.0 EMISSION SOURCES AND INVENTORIES

3.1 Natural Sources

Acrylonitrile does not occur naturally (EC, 2000; IPCS, 1983). There is no evidence that chemical reactions may lead to its formation in the atmosphere (EC, 2000).

3.2 Anthropogenic Sources

3.2.1 Industrial

According to the NPRI (NPRI, 1999), the industrial sectors contributing to acrylonitrile emissions, in order of importance, are the chemical and chemical products sector, the transportation equipment industries and the plastic product industries. The chemical and chemical products sector includes industries producing industrial organic chemicals such as plastics, resins, glycols, monomers and additives for petroleum products (NPRI, 1999). In the transportation equipment industries, the motor vehicle parts and accessories industries (steering, suspension, wheels and brakes) are responsible for acrylonitrile emissions. The plastic products industries include the foamed and expanded plastic products industries and other plastic products industries.

Table 3-1 and 3-2 indicate that, according to the NPRI database (NPRI, 1999), there are no facilities in Alberta emitting acrylonitrile. It should be noted; however, that a facility is only required to report to the NPRI if it meets all three of the following criteria (NPRI, 1999):

- the facility has more than 10 full-time employees,
- the facility manufactured, processed or used 10 tonnes or more of an NPRI substance in the calendar year,
- the facility manufactured, processed or used an NPRI substance at a concentration greater than or equal to 1% by weight.

It may be possible that acrylonitrile is emitted in Alberta in such small amounts that the facilities are not required to report to NPRI. Based on the uses reported for acrylonitrile in the previous section, it is unlikely that emissions occur from sectors not reporting to NPRI.

3.2.2 Other

Other sources of acrylonitrile identified by Environment Canada (2000) include vehicle exhaust, municipal wastewater treatment, transboundary sources and pesticide use. Cigarette smoke has also been identified as a source of acrylonitrile (Howard, 1989). Emissions of acrylonitrile from vehicles are not considered significant (EC, 2000). Acrylonitrile emissions from municipal wastewater treatment are the result of sewage sludge incineration. Only three facilities in Canada (Toronto Main, Toronto Highland Creek and Quebec) can potentially produce acrylonitrile. Emissions from these facilities have been estimated at 0.19 tonnes per year, which is not significant compared to industrial emissions. Transboundary sources are also considered insignificant due to the large distance from American sources and the small half-life of acrylonitrile in the atmosphere (EC, 2000). Since acrylonitrile is no longer used as a pesticide, emissions from this source are now considered to be zero (EC, 2000). Finally, emissions of acrylonitrile from cigarette smoke are expected to be insignificant.

Table 3-1 Total Emissions of Acrylonitrile According to NPRI, 1999 (in tonnes)

NPRI ID	Company	City	Province	Acrylonitrile Emissions (tonnes)				
				Air	Water	Land	Underground	Total
1944	Bayer Inc.	Sarnia	ON	6.860	0	0	0	6.860
3152	Oxford Automotive	Wallaceburg	ON	1.325	0	0	0	1.325
4934	ATC Chimiques/Chemicals Inc.	Drummondville	QC	0	0	0	0	0.950
2966	Waltec Plastics Inc.	Midland	ON	0	0	0	0	0.100
2969	Waltec Plastics Inc.	Midland	ON	0	0	0	0	0.100
3977	Akzo Nobel Chemicals Ltd.	Saskatoon	SK	0	0	0	0	0.100
2065	Rohm and Haas Canada Inc.	West Hill	ON	0	0	0	0	0.020
5852	Union Carbide Canada Inc.	Sarnia	ON	0	0	0	0	0.003
3146	Dow Chemical Canada Inc.	Sarnia	ON	0	0	0	0	0.002
5405	Brake Parts Canada inc.	Anjou	QC	0	0	0	0	0
				8.185	0	0	0	9.460

Table 3-2 Air Emissions of Acrylonitrile According to NPRI, 1999 (in tonnes)

NPRI ID	Company	City	Province	Air Emissions of Acrylonitrile (tonnes)					
				Stack /Point	Storage /Handling	Fugitive	Spills	Other Non-Point	Total
1944	Bayer Inc.	Sarnia	ON	1.860	0	5.000	0	0	6.860
3152	Oxford Automotive	Wallaceburg	ON	0	0	0	0	1.325	1.325
4934	ATC Chimiques/Chemicals Inc.	Drummondville	QC	0	0	0	0	0	0
2966	Waltec Plastics Inc.	Midland	ON	0	0	0	0	0	0
2969	Waltec Plastics Inc.	Midland	ON	0	0	0	0	0	0
3977	Akzo Nobel Chemicals Ltd.	Saskatoon	SK	0	0	0	0	0	0
2065	Rohm and Haas Canada Inc.	West Hill	ON	0	0	0	0	0	0
5852	Union Carbide Canada Inc.	Sarnia	ON	0	0	0	0	0	0
3146	Dow Chemical Canada Inc.	Sarnia	ON	0	0	0	0	0	0
5405	Brake Parts Canada inc.	Anjou	QC	0	0	0	0	0	0
				1.860	0	5.000	0	1.325	8.185

4.0 EFFECTS ON HUMANS AND ECOLOGICAL RECEPTORS

4.1 Humans and Animals

The primary route of exposure to acrylonitrile for the general public and animals is via inhalation. The following health summary focuses mainly on the effects associated with inhalation of acrylonitrile. It is unlikely that significant exposure to acrylonitrile would occur in the absence of an industrial source or hazardous waste facility (ATSDR, 1999).

Acrylonitrile acts acutely on the nervous system and the lungs. Chronic exposure also adversely affects the nervous and respiratory systems and has been demonstrated to be carcinogenic in a number of rat species. Results of cancer development in human studies have been inconclusive; however, due to the strong evidence of carcinogenicity in rats, acrylonitrile should be considered to be potentially carcinogenic to humans (EC, 2000; ATSDR, 1999; IARC 1999; ATSDR, 1990; US EPA 1991).

4.1.1 Overview of Chemical Disposition

Acrylonitrile is absorbed via the lungs after inhalation in animals and humans, animal gastrointestinal tract after oral exposures, and human skin after dermal application. No human oral studies or animal dermal studies were available (Rogaczewska and Pitrowski, 1969; Vogel and Kirkendall, 1984; Jakubowski et al., 1987; Pilon et al., 1988). Animal studies indicate that once absorbed, acrylonitrile is rapidly distributed throughout the body (Young et al., 1977; Ahmed and Farooqui, 1982; Ahmed et al., 1983c; Silver et al., 1987; Pilon et al., 1988). For humans and rats, the predominant route of excretion is in the urine (Young et al., 1977; Sakurai et al., 1978; Houthuijs et al., 1982; Gut et al., 1985; Jakubowski et al., 1987; Tardif et al., 1987).

The metabolism of acrylonitrile appears to be a critical step in the occurrence of adverse health effects and is responsible for the inter- and intra-species differences reported. Acrylonitrile is metabolized primarily in the liver via two different pathways. The first pathway detoxifies acrylonitrile via glutathione (GSH) metabolism. The second pathway bioactivates acrylonitrile via an oxidative pathway (cytochrome P450 2E1), producing a number of metabolites including 2-cyanoethylene oxide and cyanide (Thier et al., 2000; Léonard et al., 1999; Sumner et al., 1999; Kedderis et al., 1993; Gut et al., 1984; Abreu and Ahmed, 1980). *In vitro* studies have demonstrated that acrylonitrile is also bioactivated by cytochrome P450 enzymes in tissues other than the liver (gastric, neuronal, testicular, renal), some of which are target tissues for adverse effects (gastric and neuronal) (Ghanayem et al., 2000; Sumner et al., 1999; Abdel-Aziz et al., 1997; Mostafa et al., 1999; Subramanian and Ahmed, 1995).

The toxicity of acrylonitrile is thought to be due to the metabolic production of 2-cyanoethylene oxide and cyanide, and the depletion of glutathione (GSH) (Jiang et al, 1998; Ahmed et al., 1996). Although the mechanism of carcinogenicity of acrylonitrile has not been determined (Jiang et al., 1998; Whysner et al., 1998), 2-cyanoethylene oxide is a direct acting genotoxin and is considered the carcinogenic metabolite responsible for the production of tumors in rats after exposure to acrylonitrile (Léonard et al., 1999; Ahmed et al., 1996; Roberts et al., 1991; Guengerich, 1994). The lack of evidence for carcinogenicity in humans may be due to limited production and rapid degradation of 2-cyanoethylene oxide in humans (Léonard et al., 1999).

Species differences in metabolism may explain differences reported in adverse health effects and effective antidote therapy (Thier et al., 2000). An individual's metabolic differences (genetic variation) may also be responsible for the significant human differences reported. Thier et al. (2000) describes two cases of acute occupational exposure where one man had significant concentrations of cyanide in his blood, displayed cyanide toxicity, and responded to cyanide antidote therapy; whereas a second man with higher blood cyanide concentrations displayed no adverse health effects. Grunske (1949) reported the case of a group of people who were exposed while sleeping the night in a room previously sprayed with an acrylonitrile. One child died, some adults experienced mild eye and throat irritation, and the remaining adults reported no adverse health effects.

Ahmed et al. (1996) demonstrated that depletion of gastric GSH in rats resulted in increased oxidation of acrylonitrile and subsequent damage to the gastric DNA. Glutathione (GSH) protects cells from oxidative damage, which can result in tumourigenesis (Trush and Kensler, 1991; Suematsu et al., 1987). Jiang et al., (1998) reported oxidative stress in rat brain after sub-chronic exposure to acrylonitrile; there was reduction of GSH, decreased activity of antioxidant enzyme systems, and decreased levels of free radical scavengers. All three of these systems affected are responsible for the protection of tissue from damage, which could potentially develop into cancers. The above studies are significant as the biochemical changes occur in tissues which manifest acrylonitrile toxicity and carcinogenicity.

4.1.2 Genotoxicity and Mutagenicity

Acrylonitrile has been demonstrated to be genotoxic in a number of *in vitro* studies with enzymatic bioactivation, further indicating that acrylonitrile requires bioactivation. Examples of the genotoxic effects reported in human cells were sister chromatid exchanges, mutation, and unscheduled deoxyribonucleic acid (DNA) synthesis. Genotoxic effects in hamster cells included chromosomal aberrations, micronuclei, and sister chromatid exchanges. The genotoxic effects reported in rodent cells were mutations and DNA strand breaks (NTP, 2001; EC, 2000; Léonard et al., 1999; IARC, 1979). This is consistent with the production of 2-cyanoethylene oxide by the P450 enzymes. Acrylonitrile binds covalently to DNA (after bioactivation). It is also mutagenic in bacteria (IARC, 1999).

In vivo studies have had mixed results, some reporting increased genotoxicity with acrylonitrile exposure, and others with no significant results (NTP, 2001; EC, 2000). Workers with 15.3 years of exposure to acrylonitrile had no increased occurrence of chromosomal aberrations when compared to non-exposed workers (Theiss and Fleig, 1978).

4.1.3 Acute Effects

Acute health effects may occur after a short exposure to acrylonitrile. Acute acrylonitrile toxicity is thought to be associated with: i) the depletion of glutathione in the liver and possibly other tissues (gastric, neurological), effectively blocking the acrylonitrile detoxification pathway and resulting in; ii) elevated concentrations of metabolically produced cyanide; and, iii) the covalent binding of un-metabolized acrylonitrile to tissue proteins (Benz et al., 1997a; 1997b; Subramanian and Ahmed, 1995).

Dermal contact with liquid acrylonitrile can cause irritation, erythema, and blistering, as well as toxic and allergic dermatitis (IPCS, 1983).

Differences in adverse health effects have been observed between laboratory animal species (including cause of death) as a result of acrylonitrile exposure (ATSDR, 1990). Guinea pigs demonstrate a high tolerance to acrylonitrile toxicity and dogs, a low tolerance, with mice, rabbits, cats, and rats falling in between (NTP, 2001; ATSDR, 1990; Maltoni et al., 1977). The Agency for Toxic Substances and Disease Registry (ATSDR, 1990) summarized the results of animal and human studies reporting the effects of acute exposures to acrylonitrile. Table 4-1 lists some examples of the lowest and highest NOAELs (No Observable Adverse Effect Level) and LOAELs (Lowest Observable Adverse Effect Level) reported in the literature.

Table 4-1 Examples of NOAEL's and LOAEL's Associated with Acute Acrylonitrile Inhalation (Humans & Animal)

Effects Reported	Exposure Period	Air Concentration ppm (mg/m^3)[a]	Species	Reference
Systemic (dermal, ocular irritation) NOAEL	20-45 min.	16 (35)	Human	Wilson et al., 1948
Neurological NOAEL	8 hr	4.6 (10)	Human	Jakubowski et al., 1987
LOAEL (irritability)	20-45 min.	16 (35)	Human	Wilson et al., 1948
Death NOAEL	4 hr	30 (65)	Dog	Dudley and Neal, 1942
Death LOAEL	4 hr	65 (141)	Dog	Dudley and Neal, 1942
Death NOAEL	4 hr	275 (598)	Cat	Dudley and Neal, 1942
Death LOAEL	4 hr	600 (1,304)	Cat	Dudley and Neal, 1942
Systemic (hepatic, renal, and respiratory) NOAEL (hepatic)	12 hr	26-229 (56-498)	Rat	Gut et al., 1984; 1985; Rouisse, et al., 1986
Systemic (hepatic, renal, and respiratory) NOAEL (resp.&renal)	5d 8hr/d	26 –229 (56-498)	Rat	Gut et al., 1984; 1985; Rouisse, et al., 1986
Neurological NOAEL	4 hr	65 (141)	Monkey	Dudley and Neal, 1942
Developmental NOAEL	10d 6hr/d	40 (87)	Rat	Murray et al., 1978
Developmental LOAEL	10d 6hr/d	80 (174)	Rat	Murray et al., 1978

[a] The following conversion factor and assumptions were used: mg/m^3 x 24.45/MW =ppm; MW=53.06, air at 25°C and 101.3 kPa (760mmHg) (Plog et al., 1996).

4.1.3.1 Acute Adverse Effects in Experimental Animals

Acute adverse health effects observed include: salivation, lacrimation (tearing of the eyes), diarrhea, gastrointestinal injury (liver necrosis and hemorrhagic gastric bleeding of the fore stomach), seizures, and death (EC, 2000; Thier et al., 2000; Benz et al., 1997a; Subramanian and Ahmed, 1995; Buchter et al., 1984). Initial adverse health effects are usually cholineric: miosis (excessive constriction of the pupil of the eye), chromodacryorrhea (blood from the eyes), lacrimation, salivation, nasal discharge, vasodilation, vomiting, and diarrhea; these can be followed by typical cyanide-like effects: depression, rapid and shallow breathing, apnea, convulsions, and death (NTP, 2001; EC, 2000; Thier et al. 2000, IPCS, 1983).

Repeated short-term exposures have been reported to cause changes in biochemical parameters and body weight (EC, 2000).

4.1.3.2 Acute Adverse Health Effects In Humans

In humans, the majority of adverse health effects have been reported after accidental exposure via inhalation and/or skin contact. The effects include: irritation (eye and throat); central nervous system (CNS) effects (headaches, nausea, vomiting, somnolence, limb weakness, dizziness, impaired judgment, convulsions and unconsciousness); and, death (Thier et al., 2000; Baxter, 1979; Grunske, 1949). Although cardiovascular effects have not been reported in animal studies, one case of a child exhibiting tachycardia was reported after inhalation of acrylonitrile. It could not; however, be concluded that the tachycardia was due to acrylonitrile exposure (Grunske, 1949).

The CNS effects of acrylonitrile are likely associated with exposure to cyanide, a metabolic product of acrylonitrile oxidation. Cyanide levels in the blood of exposed persons were elevated and much higher than acrylonitrile concentrations (EC, 2000; ATSDR, 1990; IPCS, 1983). In addition, treatment of exposed persons with the typical antidote for cyanide toxicity has been successful (Thier et al., 2000).

Significant individual differences in response to acrylonitrile exposure have been reported with some individuals demonstrating significant adverse health effects and others showing no evidence of adverse health effects at all. One report (Grunske, 1949) indicates that children may be significantly more sensitive to acrylonitrile exposure than adults when a child died after spending the night in a room previously sprayed with an acrylonitrile. Some of the adults who slept in the same room experienced only mild eye and throat irritation; the remaining adults reported no adverse health effects. This may indicate that children are more sensitive than adults, or that the affected child was more susceptible due to individual (genetic) metabolic differences.

Estimation of human exposure dose is very difficult because exposures were often accidental and an air concentration could not be determined.

4.1.4 Chronic Effects

The ATSDR (1990) summarized the results of animal and human studies reporting the non-carcinogenic and carcinogenic effects of chronic exposures to acrylonitrile. Specific exposure

concentrations associated with adverse human health effects were not available, therefore, NOAEL's and LOAEL's could not be determined. However, effects have been reported in humans after long-term exposures have been known to occur (see Section 4.1.4.2 below). Examples of animal lowest and highest NOAELs (No Observable Adverse Effect Level) and LOAELs (Lowest Observable Adverse Effect Level) were available (see Table 4-2).

Table 4-2 Examples of NOAEL's and LOAEL's Associated with Chronic Acrylonitrile Inhalation in Animals

Effects Reported	Air Concentration ppm(mg/m^3)[a]	Species	Reference
Death LOAEL	20 (43)	Rats	Quast et al., 1980a
Systemic NOAEL respiratory	20 (43)	Rats	Quast et al., 1980a
Systemic NOAEL hepatic	80 (174)	Rats	Quast et al., 1980a
Dermal, Ocular NOAEL	80 (174)	Rats	Quast et al., 1980a
Neurological and Electrophysiological NOAEL	25 (54)	Rats	Gagnaire et al., 1998
Neurological and Electrophysiological LOAEL	50 (109)	Rats	Gagnaire et al., 1998
Developmental[1] short tail and missing vertebrae NOAEL	40 (87)	Rats	Murray et al., 1978
Developmental[1] short tail and missing vertebrae LOAEL	80 (174)	Rats	Murray et al., 1978
Cancer NOAEL (brain and spinal cord; Zymbal gland, small intestine and tongue, mammary glands).	20 (43)	Rat	Quast et al., 1980a
Cancer LOAEL's (mammary gland (females); Zymbal gland (males) extrahepatic, encephalic)	60 (130)	Rat	Maltoni et al., 1988
Cancer LOAEL (brain and spinal cord; Zymbal gland, small intestine and tongue (males) mammary glands (females).	80 (174)	Rat	Quast et al., 1980a

[a] The following conversion factor and assumptions were used: mg/m^3 x 24.45/MW =ppm; MW=53.06, air at 25°C and 101.3 kPa (760mmHg) (Plog et al., 1996).

[1] NB: Effects reported only at high doses and with maternal toxicity (EC, 2000; Saillenfait et al.1993; ATSDR, 1990; Murray et al., 1978)

4.1.4.1 Animal Chronic Effects

4.1.4.1.1 Non-Carcinogenic Effects

Chronic acrylonitrile exposure produces the same cholinergic effects described in the Acute Effects section: salivation, lacrimation, chromodacryorrhea, miosis or diarrhea. Other neurological effects reported were changes in peripheral nerve function and hind limb weakness (Gagnaire et al., 1998). Some of these effects were transient, others became more pronounced with continued exposures. In addition, rodent species have demonstrated: inflammation of nasal turbinates (inhalation studies only); decreases in body weight gain; hematological changes; increases in liver and kidney weight; and, at high exposure doses, increased morbidity.

Reproductive effects in rodents (degeneration of the germinal epithelium and seminiferous tubules, decreased sperm count and decreased sperm motility) have also been associated with acrylonitrile exposure (Ahmed et al., 1993b; Tandon et al., 1988). *In vitro* studies (rat embryos) indicate acrylonitrile is embryotoxic, particularly when liver microsomal enzymes were added to the test system (Sallenfait and Sabaté, 2000). Developmental effects reported may be due to maternal metabolically produced cyanide (Léonard et al., 1999; Willhite, 1983; Willhite et al., 1981). As in the other exposure/effects studies described above, the reproductive and embryotoxic effects reported appear to be due primarily to metabolites of acrylonitrile. Fetotoxic and teratogenic effects have been reported in animals (rats and hamsters); however, only at high doses and with maternal toxicity (EC, 2000; Saillenfait et al., 1993; ATSDR, 1990; Murray et al., 1978).

Systemic immunosuppressive effects have been reported in rats and mice (Hamada et al., 1998; Ahmed et al., 1993a). Hamada et al. (1998) demonstrated immunosuppression locally in the gastrointestinal tract of exposed mice.

4.1.4.1.2 Carcinogenic Effects

Inhalation of acrylonitrile has been demonstrated to be carcinogenic in rats. Chronic inhalation produced tumors in the brain, Zymbal gland, mammary glands (females), small intestines (males), and tongue (males) (Quast et al., 1980a; Maltoni et al., 1988). In some studies, ingestion of acrylonitrile was also carcinogenic in rats (BioDynamics, 1980a;b;c; Quast et al., 1980b; Maltoni et al., 1977); however, in other studies, cancers were not associated with ingestion (Maltoni et al., 1988). The only other study of carcinogenicity of acrylonitrile in an animal species besides rats was NTP (2001), which reported carcinogenicity in mice after exposure via gavage. However, this report has not completed the peer review process and is only available as a board draft, thus, details of the results have not been finalized.

4.1.4.2 Human Chronic Effects

Long-term exposure to acrylonitrile has been reported to affect a variety of the body's systems. Long-term human exposure has occurred in occupational settings only, therefore, a number of confounding factors (i.e., exposure to other chemicals and lifestyle factors, specifically, smoking) exist when trying to document the potential effects of exposure to acrylonitrile. Exposure doses could not be reliably determined and clinical findings reported in some studies could not be identified in others.

4.1.4.2.1 Non-Carcinogenic Effects

The following is a summary of non-carcinogenic effects reported in workers from facilities where occupational exposure to acrylonitrile (primarily by inhalation) was known to occur.

Clinical observations: Poor health, headache, general fatigue, irritability, depression, anorexia, nausea, vomiting, heartburn, chest pains, shortness of breath, nose bleeds, insomnia, excessive perspiration, skin irritation, and weakness (Muto et al., 1992; IPCS, 1983; Sakurai et al., 1978; Zotova, 1975; Wilson, 1944; Wilson et al., 1948).

Haematological: Some exposed workers were reported to present with anemia; reduced haemoglobin level, erythrocyte count, leukocyte count, and percentage of neutrophiles; and an increased percentage of lymphocyctes and plasma iron (Zotova, 1975). In a different study of workers exposed 10 to 13 years, no hematological changes were observed (Sakurai et al., 1978).

Immunological: Decreased T-lymphocyte functional activity (IPCS, 1983).

Neurological: Nausea, vomiting, headache, vertigo, depression, decreased blood pressure, and increased sweating (Muto et al., 1992; IPCS, 1983; Baxter, 1979; Sakurai et al., 1978; Zotova, 1975; Wilson, 1944; Wilson et al., 1948).

Gastrointestinal: Gastritis and colitis (Enikeeva et al., 1976).

Dermal: Irritation, and allergic dermatitis and sensitization (Stamova et al., 1976; Spassovski, 1976; Antonev and Rogailin, 1970).

No conclusive human reproductive or developmental effects data were identified. However, acrylonitrile has been reported to interfere with testosterone synthesis and/or secretion in men exposed occupationally (Ivanescu et al., 1990).

4.1.4.2.2 Carcinogenic Effects:

Early studies of occupational exposure reported potential association between acrylonitrile exposure and increases in lung cancers (Thiess et al., 1980; Denzell and Monson, 1982; O'Berg, 1980) and "all cancers" (Zhou and Wang, 1991). However, more recent assessment of human exposure data (in some cases the same exposure data) do not confirm these results (Blair et al., 1998; Collins and Aquavella, 1998; Marsh et al., 1998; Swaen et al., 1998; Wood et al., 1998; Felter and Dollarhide, 1997; Benn and Taylor, 1997; Mastrangelo et al., 1993; Swaen et al.1992; Collins et al., 1989; Chen et al., 1987). This may be because of individual differences, the small number of deaths reported in the human data, and/or the potential occurrence of cancers, which are difficult to detect in small data sets (stomach, brain, prostate, lymphatic/hematopoietic) (EC, 2000; Strother et al., 1998; ATSDR, 1990; O'Berg et al., 1985).

The study of O'Berg (1980) has been described as the most reliable human study of acrylonitrile exposure. This study identified a potential increase in lung cancer; however, the statistical significance was not apparent in a 1985 follow up study of the same population (O'Berg et al., 1985). Re-evaluation of a data set indicated that there might be an increased incidence of lung cancer with increased exposure to acrylonitrile (Marsh et al., 1998). An increase in prostate

cancer (not mortality due to prostate cancer) has also been reported in some of the re-evaluations of the available epidemiological data (Wood et al., 1998; O'Berg et al., 1985; Chen et al., 1987); however, others did not support these findings (Blair et al., 1998; Marsh et al., 1998; Swaen et al., 1992; Collins et al., 1989).

Although inhalation of acrylonitrile has been demonstrated to be carcinogenic in rats in controlled laboratory studies, reports on human exposures have not been able to conclusively demonstrate an increased cancer incidence as a result of acrylonitrile exposure (Environmental Canada/Health Canada 2000; ATSDR, 1990; Blair et al., 1998; Collins and Aquavella, 1998; Marsh et al., 1998).

4.1.5 Summary

Inhalation of acrylonitrile can result in significant adverse health effects both acutely and chronically. Chronic exposure in rats is carcinogenic. In humans; however, chronic exposure has not conclusively been shown to result in cancer.

Acrylonitrile is bioactivated to produce two metabolic toxins: 2-cyanoethylene oxide and cyanide. Toxicity associated with inhalation of acrylonitrile is thought to be due to the production of cyanide, and tissue depletion of Glutathione (GSH). The carcinogenic effects seen in rats may be associated with the production of 2-cyanoethylene oxide, a genotoxin.

4.2 Vegetation

The pathways of entry of chemical constituents into terrestrial plants are through the air, soil or by both mechanisms. No published literature could be found on the direct effects (through the atmosphere) of acrylonitrile on terrestrial plants.

In the context of soil serving as a medium for the entry of acrylonitrile into plants, concentrations of up to 5000 mg/L did not appear to be toxic to soil bacteria and were readily degraded by *Corynebacterium* sp and *Arthrobacter* sp (Narayanaswamy et al. 1990; Wenzhong et al. 1991;).

Tong and Hongjun (1997) showed that the aquatic plant, duckweed (*Lemna minor*) could be used as an effective bioassay for acrylonitrile. After a 96-hour artificial or experimental exposure, relative plant growth rate was inhibited by 50% at a concentration of 27.08 mg/L.

5.0 AIR SAMPLING AND ANALYTICAL METHODS

5.1 Reference Methods

Air sampling and monitoring methods for acrylonitrile used in practice by established agencies are reported. In general, standard air monitoring methods for acrylonitrile are based on volumetric sampling, canister sampling or pump-and-tube sampling approaches. Widely employed and accepted referenced air monitoring methods and technologies for acrylonitrile have been developed, tested and reported by the United States Environmental Protection Agency (US EPA), National Institute of Occupational Safety and Health (NIOSH), and Occupational Safety and Health Administration (OSHA). Refer to Table 5-1 for a description of individual method advantages and disadvantages.

5.1.1 US EPA Compendium Method TO-2

US EPA Compendium Method TO-2 has developed a number of methodologies suitable for sampling ambient air for trace-level concentrations of acrylonitrile. US EPA Compendium Method TO-2 describes the determination of volatile organic compounds (VOCs) (including acrylonitrile) in ambient air by carbon molecular sieve (CMS) adsorption and gas chromatography/mass spectrometry (GC/MS) (US EPA, 1999). The advantages of this method include: trace levels of VOCs are collected and concentrated on sorbent material, efficient collection of polar compounds, wide range of applications, highly volatile compounds are adsorbed, and easy to use in the field. Disadvantages of this method include: some trace levels of organic species are difficult to recover from the sorbent, structural isomers are common interferences, water is collected and can de-activate adsorption sites, and thermal desorption of some compounds may be difficult.

In this method, ambient air is drawn through a cartridge containing 0.4 grams (g) of a CMS adsorbent. A sampling pump system capable of accurately and precisely drawing airflow at a rate of 10 to 500 milliliters per minute (ml/min) through the CMS cartridge is used. VOCs are captured on the adsorbent while major inorganic atmospheric constituents pass through (or are only partially retained). After sampling, the cartridge is returned to the laboratory for analysis. Prior to analysis, the cartridge is purged with 2 to 3 liters (L) of pure, dry air (in the same direction as the sample flow) to remove adsorbed moisture. For analysis, the cartridge is heated to 350 to 400°C (under helium purge) and the desorbed organic compounds are collected in a specially designed cryogenic trap. The collected organics are then flash evaporated onto a capillary column GC/MS system (held at –70°C). The individual components are subsequently identified and quantified during a temperature programmed chromatographic run.

5.1.2 US EPA Compendium Method TO-15A

US EPA Compendium Method TO-15A describes the determination of VOCs (including acrylonitrile) in air collected in specially prepared canisters and analyzed by GC/MS (US EPA, 1999). The advantages of this method include: incorporates a multisorbent/dry purge technique or equivalent for water management thereby addressing a more extensive set of compounds, establishes method performance criteria for acceptance of data, provides enhanced provisions for quality control, and unique water management approach allows analysis for polar VOCs.

Disadvantages of this method are it requires expensive analytical equipment and a high level of operator skill to perform.

In this method, the ambient atmosphere is sampled by introduction of 6 L of air into a specially prepared stainless steel canister (SUMMA or equivalent) over an appropriate time and rate. Both subatmospheric pressure and pressurized sampling modes make use of an initially evacuated canister. A pump ventilated sampling line is used during sample collection with most commercially available samplers. Pressurized sampling requires an additional pump to provide positive pressure to the sample canister. A sample of air is drawn through a sampling train comprised of components that regulate the rate and duration of sampling into the pre-evacuated and passivated canister. After the air is collected the canister valve is closed, an identification tag is attached to the canister, and the canister is transported to the laboratory for analysis. Upon receipt at the laboratory the canister tag data is recorded and the canister is stored until analysis.

To analyze the sample a known volume of sample is directed from the canister through a solid multisorbent concentrator. A portion of the water vapour in the sample breaks through the concentrator during sampling to a degree depending on the multisorbent composition, duration of sampling, and other factors. Dry purging the concentrator with helium while retaining target compounds can further reduce water content of the sample. After the concentration and drying steps are completed, the VOCs are thermally desorbed, entrained in a carrier gas stream, and then focused in a small volume by trapping on a reduced temperature trap or a small volume multisorbent trap. The sample is then released by thermal desorption and carried onto a gas chromatographic column for separation.

The analytical strategy for US EPA Compendium Method TO-15A involves using a high-resolution gas chromatograph (GC) coupled to a mass spectrometer (MS). If the MS is a linear quadrupole system, it is operated either by continuously scanning a wide range of mass to charge ratios (SCAN mode) or by monitoring select ion monitoring mode (SIM) of compounds on the target list. If the MS is based on a standard ion trap design, only a scanning mode is used. Mass spectra for individual peaks in the total ion chromatogram are examined with respect to fragmentation pattern of ions corresponding to various VOCs including the intensity of primary and secondary ions. The fragmentation pattern is compared with stored spectra taken under similar conditions, in order to identify the compound.

For any given compound, the intensity of the primary fragment is compared with the system response to the primary fragment for known amounts of the compound. This establishes the compound concentration that exists in the sample. This method applies to ambient concentrations of VOCs above 0.5 parts per billion by volume (ppbv) and typically requires VOC enrichment by concentrating up to 1 L of a sample volume. The VOC concentration range for ambient air in many cases includes the concentration at which continuous exposure over a lifetime is estimated to constitute a 10^{-6} or higher lifetime risk of developing cancer in humans.

5.1.3 US EPA Compendium Method TO-17

US EPA Compendium Method TO-17 describes the determination of VOCs (including acrylonitrile) in ambient air using active sampling onto sorbent tubes (US EPA, 1999). This method is an alternative to the canister-based sampling and analysis methods presented in

Compendium Method TO-15A and to the sorbent-based method presented in Compendium Method TO-2. The advantages of this method include: placement of the sorbent as the first element minimizes contamination from other sample train components, large selection of sorbents to match with target analyte list, includes polar VOCs, better water management using hydrophobic sorbents, large database and proven technology, and size and cost advantages in sampling equipment. Disadvantages of this method include: distributed volume pairs required for quality assurance, rigorous clean-up of sorbent required, no possibility of multiple analysis, must purchase thermal desorption unit for analysis, desorption of some VOCs is difficult, and contamination of adsorbent can be a problem.

In this method, ambient air is actively drawn through a multi-bed sorbent tube where VOCs are trapped. The selection of tube and sorbent depends on the VOC in question. In the case of acrylonitrile, suitable commercially available sorbent tubes include: Porapak-N, Carbopack-B or Combination Tubes 1, 2 or 3. A sampling apparatus with accommodations for two sampling tubes capable of independent control of sampling rates is used. For acrylonitrile, sampling rates of 16.7 ml/min and 66.7 ml/min are used to collect 1 and 4 L total sample volumes over a 1-hour period, respectively. The sorbent tube is returned to the laboratory where it is thermally desorbed and subsequently analyzed by GC/MS or other methods. This method applies to ambient concentrations of VOCs of 0.5 to 25 ppbv.

5.1.4 US EPA Method 0030

The US EPA has also developed a number of methodologies suitable for sampling gaseous emissions from a wide variety of stationary sources for the determination of trace-level concentrations of acrylonitrile. US EPA Method 0030 - Volatile Organic Sampling Train (VOST) describes the methodology for the collection of volatile principle organic hazardous constituents (POHCs) (including acrylonitrile) from the stack gas effluents of hazardous waste incinerators (US EPA, 1986). For the purpose of definition, volatile POHCs are those POHCs with boiling points less than 100°C. This method employs a 20 L sample of effluent gas containing volatile POHCs that is withdrawn from a gaseous effluent source at a flow rate of 1 liter per minute (L/min), using a glass-lined probe and a VOST.

The gas stream is cooled to 20°C by passage through a water-cooled condenser and volatile POHCs are collected on a pair of sorbent resin traps. Liquid condensate is collected in an impinger placed between the two resin traps. The first resin trap (front trap) contains approximately 1.6 g of Tenax and the second trap (back trap) contains approximately 1 g each of Tenax and a petroleum-based charcoal, 3:1 by volume. A total of six pairs of sorbent traps may be used to collect volatile POHCs from the effluent gas stream. Analysis of the traps is carried out by thermal desorption purge-and-trap by GC/MS. The VOST is designed to be operated at 1 L/min with traps being replaced every 20 minutes for a total sampling time of 2 hours. Traps may be analyzed separately or combined onto one trap to improve detection limit. The target detection limit of this method is 0.1 to 100 micrograms per cubic meter ($\mu g/m^3$) for selected POHCs collected on a set of sorbent traps using a total sample volume of 20 L or less.

5.1.5 EPA Method 0031

US EPA Method 0031 - Sampling Method for Volatile Organic Compounds (SMVOC) may be used to measure VOCs that have a boiling point between –15°C and 121°C (including acrylonitrile) (US EPA, 1996). This method employs a sampling module and meter box to withdraw a 20 L sample of effluent gas containing volatile organic compounds from a stationary source at a flow rate of 1 L/min, using a glass-lined probe heated to 130°C and a sampling method for SMVOC train.

The gas stream is cooled to 20°C by passage through a water-cooled condenser and volatile organic compounds are collected on a set of sorbent traps (Tenax-GC/Tenax-GC/Anasorb-747). Liquid condensate is collected in an impinger placed between the two Tenax-GC traps and the Anasorb-747 trap. The first and second traps contain approximately 1.6 g of Tenax-GC each and the third trap (back trap) contains approximately 5.0 g of Anasorb-747. A total number of sorbent sets to encompass a total sampling time of 2 hours are collected (i.e. if a sampling rate of 1 L/min for 20 minutes is used, a total of six sorbent tube sets will be collected in 2 hours of sampling). Analysis of the traps is carried out by thermal desorption purge-and-trap by GC/MS. The target detection limit of this method is 0.1 to 100 $\mu g/m^3$ for selected VOCs collected on a set of sorbent traps using a total sample volume of 20 L or less.

5.1.6 NIOSH Method 1604

In addition to the referenced air monitoring methods for acrylonitrile developed by the US EPA, both the NIOSH and OSHA have also developed methods for acrylonitrile that are suitable for occupational, personal and area monitoring. The current methodology used by the NIOSH to determine acrylonitrile in air (NIOSH Method 1604) consists of collecting acrylonitrile on charcoal, desorbing with a solution of acetone in carbon disulfide, and analyzing by GC using a flame ionization detector (FID) (NIOSH, 1994). Sampling is conducted by drawing air through standard size coconut shell charcoal absorbent tubes (100 milligrams (mg) in the front section and 50 mg in the back section) using a personal sampling pump calibrated to within 5% of the recommended flow rate with a sampling tube in line. The suggested flow rate is between 0.01 and 0.2 L/min and the recommended volume collected is between 3.5 and 20 L. The working acrylonitrile concentration range for this method is 0.7 to 46 parts per million by volume (ppmv) (1.5 to 100 milligrams per cubic meter (mg/m^3)) for a 10 L sample.

5.1.7 *OSHA Method 37*

The current methodology used by the OSHA to determine acrylonitrile in air (OSHA Method 37) was developed to be convenient, sensitive and accurate (OSHA, 1982). This method consists of collecting acrylonitrile on charcoal, desorbing with acetone containing an internal standard, and analyzing by GC using a nitrogen/phosphorous detector (NPD). It is similar to the NIOSH method except that NPD is used instead of a FID. OSHA has chosen NPD as the analytical technique because the sensitivity and selectiveness of FID for acrylonitrile is lower than that of NDP. Advantages of this method include: charcoal tubes are readily available, the samples may be analyzed more than once if necessary, the method is rapid and precise, and the NPD is selective and sensitive. A disadvantage of this procedure is that acrylonitrile does not have constant desorption at loadings below 40 micrograms (µg) for the 100 mg charcoal section and 16 µg for the 50 mg section.

Sampling is conducted by drawing air through standard size coconut shell charcoal absorbent tubes (100 mg in the front section and 50 mg in the back section) using a personal sampling pump calibrated to within 5% of the recommended flow rate with a sampling tube in line. The suggested flow rate is 0.2 L/min and the recommended volume collected is 20 L. The reliable detection limit of the overall procedure is 0.51 µg per sample (0.01 ppmv or 0.026 mg/m^3) for a 20 L air sample. This is the amount of acrylonitrile spiked on a charcoal tube that allows recovery of an amount equivalent to the detection limit of the analytical procedure. The reliable quantitation limit is 13.2 µg per sample (0.3 ppmv or 0.66 mg/ m^3). This is the smallest amount of acrylonitrile that can be quantified within the requirements of 75% recovery and 95% confidence limits of ± 25%.

5.2 Alternative, Emerging Technologies

In general, most non-standard methods and technologies are variations or modifications of those referenced methods previously mentioned (Campbell and Moore, 1979; Borders et al., 1986; NIOSH, 1994; Mastrogiacomo et al., 1998). However, a number of unique methods and technologies have been described (IPCS, 1983; Brown and Wright, 1994; Harper, 1994; Levin and Lindahl, 1994; Uchiyama and Hasegawa, 2000). These unique methods and technologies include: passive/diffusive sampling, direct and continuous sampling, and long-term sampling.

The most notable variations or modifications of referenced methods for the collection and analysis of volatile organic compounds (VOCs) (including acrylonitrile) involve alternative types of sorbents to be used in conjunction with the accepted pump-and-tube sampling approaches. Of the alternative sorbents, porous polymers or silica gel are the most common. In addition, a new adsorbent high-surface-area graphitized carbon black (HSGCB) was found to be an interesting alternative to the usual sorbents used for sampling VOCs such as acrylonitrile (Mastrogiacomo et al., 1998).

A number of different analytical techniques have also been recommended. Borders et al. (1986) suggest using thermal desorption as an alternative to solvent desorption for the determination of acrylonitrile in air. Samples are collected by pulling air through a stainless steel tube containing thermally stable sorbents. The tube is heated and the absorbed compounds are purged directly into a gas chromatograph (total injection thermal desorption technique) or into an intermediate volume, which is subsequently analyzed by gas chromatography with mass spectrometry (GC/MS), instead of removing the sorbent and extracting with a solvent for analysis. Thermal desorption eliminates the use of solvents and other handling operations and is more sensitive than solvent desorption techniques. Another method for detecting acrylonitrile in air previously used by the NIOSH uses methanol instead of other solvents for desorption and gas chromatography (GC) with a flame ionization detector (FID) for analysis (NIOSH, 1994). Finally, Campbell and Moore (1979) describe a method for detecting acrylonitrile in air that uses gas chromatography with a nitrogen/phosphorous detector (GC/NPD) for analysis and methanol to desorb the charcoal but with sonication to increase the desorption efficiency.

An increasing number of passive or diffusive samplers (gas badges or diffusion tubes) have been developed to determine concentrations of acrylonitrile in air as an alternative to the pump-and-tube techniques. For diffusive sampling, the same collecting media (sorbent) as for pumped

sampling can be used. The advantages of these samplers are that there are no moving parts to break down, regular flow calibration is unnecessary, and no bulky, expensive pumps are required. The badge or tube is exposed to ambient conditions for a set period of time (usually a longer period than for active pump sampling) and then analyzed by GC/FID or another similar analytical method (Brown and Wright, 1994; Levin and Lindahl, 1994).

The World Health Organization (IPCS, 1983) describes a passive dosimeter (badge) in which acrylonitrile is adsorbed on a porous polymer (Porapak-N) contained in a removable element and determined by thermal desorption gas chromatography. It can be used satisfactorily for determining acrylonitrile concentrations in air under a range of atmospheric conditions, when working to a control limit of 8.7 milligrams per cubic meters (mg/m^3) (4 parts per million by volume (ppmv)) but, at a concentration of 4.4 mg/m^3 (2 ppmv), a 40% error has been reported. These devices are now considered to be as reliable as the more conventional pump-and-tube techniques.

Direct, continuously recording gas chromatographic and other analytical methods have been developed and utilized for monitoring atmospheric concentrations of acrylonitrile. These devises reportedly detect acrylonitrile below 1.1 mg/m^3 (0.5 ppmv) (IPCS, 1983). Direct-reading instruments with fast response times offer the best solution for monitoring average concentrations accurately, but many are bulky and expensive. In addition, many instruments are either insufficiently sensitive or selective in their measurements (Harper, 1994).

In many cases, monitoring methods need to be designed which accurately estimate the annual or monthly average ambient concentrations of VOCs to understand the overall risk to public health. Instant or short-term sampling can only be used for determination of a momentary value for VOC concentration and cannot be considered representative of the annual or monthly average when actual atmospheric concentrations vary so significantly over time. Uchiyama and Hasegawa (2000) have investigated a long-term, low-flow monitoring method for the measurement of VOCs (including acrylonitrile) in ambient air. Long-term, non-stop sampling periods of 4-weeks, 7-days, and 24-hours were evaluated simultaneously, using mass flow controllers set to 0.5, 2.0, and 14 milliliters per minute (ml/min), respectively. Ambient air was drawn through a multisorbent sampling tube packed with Carbotrap C, Carbotrap B, and Carboxen 1000. Adsorbates were introduced into a GC/MS using a thermal desorption cold trap injector and determined according to the US EPA Compendium Method TO-15A (US EPA, 1999). The study concluded long-term ambient concentrations of almost all VOCs (including acrylonitrile) could be accurately monitored by this technique using 7-day or 28-day sampling periods.

Table 5-1 Method Advantages and Disadvantages

Method	Advantages	Disadvantages
U.S. EPA Compendium Method TO-2	Trace levels of VOCs are collected Efficient collection of polar VOCs Wide range of applications Highly volatile compounds are adsorbed Easy to use in the field	Some species difficult to recover Structural isomers are common interferences Water is collected and can de-activate adsorption sites Thermal desorption of some compounds difficult
U.S. EPA Compendium Method TO-15A	Addresses a large set of compounds Establishes method performance criteria for acceptance of data Provides quality control provisions Allows analysis for polar VOCs	Requires expensive analytical equipment Requires high level of operator skill
U.S. EPA Compendium Method TO-17	Minimizes contamination Large selection of sorbents available Allows analysis for polar VOCs Better water management Large database and proven technology Equipment size and cost advantage	Distributed volume pairs required for quality assurance Rigorous clean-up required No possibility of multiple analysis Require thermal desorption unit Desorption of some VOCs difficult Contamination of adsorbent possible
U.S. EPA Method 0030	NA	NA
U.S. EPA Method 0031	NA	NA
NIOSH Method 1604	NA	NA
OSHA Method 37	Equipment readily available Can perform analysis more than once Rapid and precise Method is selective and sensitive	Acrylonitrile does not have a constant desorption at low loadings with this procedure
Alternative sorbent types	NA	NA
Alternative analytical techniques	Some eliminate the use of solvents and other handling operations Some are more sensitive Some increase desorption efficiency	NA
Passive samplers	No moving parts to break down Regular flow calibration unnecessary No bulky, expensive pumps required As reliable as conventional methods	Only reliable at higher ambient concentrations
Direct and continuous samplers	Reliable at low concentrations Fast response times Measure average concentrations accurately	Bulky and expensive equipment required Many are insufficiently sensitive or selective in their measurements
Long-term samplers	Long-term concentrations can be accurately measured	Experimental

NA denotes not available.

6.0 AMBIENT GUIDELINES

Current and/or recommended and proposed ambient guidelines from jurisdictions in Canada, United States and elsewhere (other than in Alberta) were reviewed for acrylonitrile. These guidelines are presented in Table 6-1. In general, all jurisdictions have common uses for their guidelines in practice. These uses may include:

- reviewing permit applications for sources that emit air pollutants to the atmosphere,
- investigating accidental releases or community complaints about adverse air quality for the purpose of determining follow-up or enforcement activity,
- determining whether to implement temporary emission control actions under persistent adverse air quality conditions of a short-term nature.

The three principal approaches by which guidelines are developed for substances like acrylonitrile include:

- Using an occupational exposure level (OEL) and dividing it by safety or adjustment factors. The most common OEL used by state agencies is the 8-hour threshold limit value (TLV) of 4,300 $\mu g/m^3$ (2 ppm) adopted by the American Conference of Governmental Industrial Hygienists (ACGIH). The safety or adjustment factors are intended to account for issues such as: differences between eight-hour exposures in the workplace and continuous 24-hour environmental exposures, increased susceptibility of some people in the general population versus the relatively healthy worker, and uncertainty in the margin of safety provided in an occupational exposure limit.
- Using non-carcinogenic risk assessment procedures. A no observed adverse effect level (NOAEL) – or lowest observed adverse effect level (LOAEL) if a NOAEL is unavailable – from a suitable animal or human study is used. It is then divided by a series of adjustment factors. The adjustment factors are intended to account for issues such as: differences between animals and humans, sensitivity of high risk individuals, use of a LOAEL instead of a NOAEL, and for extrapolation from less-than-lifetime exposures to chronic exposure.
- Using carcinogenic risk assessment procedures. Pre-existing cancer risk assessment information summarized by others (e.g. US EPA Integrated Risk Information System summary data) are used to establish ambient air levels based on acceptable levels of increased lifetime cancer risk, such as 1 in 100,000 (10^{-5}).

For the most part, the guidelines in Table 6-1 are derived based on injury of nasal respiratory epithelium in rats exposed through inhalation (non-carcinogenic effect endpoint) or epidemiological study of respiratory cancer from inhalation in the workplace (carcinogenic effect endpoint). Almost all of the guidelines listed in Table 6-1 are for chronic exposure durations. Further information on how these guidelines were developed and how they are used in practice is provided in Appendix A.

6.1 Canada

Health Canada and Environment Canada have listed acrylonitrile as a priority chemical for risk assessment under the *Canadian Environmental Protection Act* (CEPA, 1999). These agencies considered tumour formation as the critical health effects endpoint via inhalation exposure in the development of tumourigenic concentration (TC_{05}) values for the tumour-causing effect of acrylonitrile. A tumorigenic concentration (TC_{05}) is the concentration generally in air associated with a 5% increase in incidence or mortality due to tumours. TC_{05}s divided by a suitable margin provide a benchmark against which the adequacy of exposure to ambient or indoor air can be judged, with respect to potential carcinogenicity. These agencies applied safety factors of 5,000 and 50,000 to the TC_{05} to derive risk concentrations of 1.2 and 0.12 $\mu g/m^3$ (0.6 and 0.06 ppb), representing excess cancer risks of 1 in 100,000 and 1 in 1,000,000, respectively.

Ontario MOE (2001) used these data in developing ambient air quality criteria (AAQC) listed in Table 6-1. AAQC represent human health or environmental effect-based values, and are normally set at a level not expected to cause adverse effects based on continuous exposure. As such, economic factors, such as technical feasibility and costs, are not explicitly considered when establishing AAQCs. On the other hand, emission sources in Ontario are required to comply with the Point of Impingement (POI) standards, and the development of POI standards does take into account economic and other factors.

6.2 United States

The US Agency for Toxic Substances and Disease Registry developed an acute inhalation minimum risk level (MRL) applicable for 1 to 14-day exposure durations (ATSDR, 1990). Absence of neurological effects in a study with humans volunteers exposed to acrylonitrile was used to derive the chronic inhalation MRL of 217 $\mu g/m^3$ (100 ppb). MRLs are intended to serve as a screening tool to help public health professionals decide where to look more closely.

The US EPA developed a chronic inhalation reference concentration (RfC) of 2 $\mu g/m^3$ (0.9 ppb) (US EPA, 2001). The RfC is based on a two-year inhalation exposure study to rats and a LOAEL 20 ppm for injury to the nasal respiratory epithelium in rats. US EPA used data from an epidemiological study of respiratory cancer from inhalation in the workplace to derive an inhalation unit risk of 6.8E-05 per ($\mu g/m^3$). The RfC and inhalation unit risk are intended for use by US EPA staff in risk assessments, decision-making and regulatory activities.

In general, most of the US agencies reviewed have adopted or derived their values from the US EPA RfC (2 $\mu g/m^3$ or 0.9 ppb) and/or inhalation unit risk (6.8E-05 per $\mu g/m^3$). California Environmental Protection Agency uses the same principal data from an epidemiological study of respiratory cancer from inhalation in the workplace to derive an inhalation unit risk of 2.9E-04 per ($\mu g/m^3$) (Cal EPA, 1999).

Only three state agencies listed in Table 6-1 – New Hampshire, Oklahoma, and Texas – use the ACGIH 8-hour TLV (4,300 $\mu g/m^3$ or 2 ppm) in development of various ambient guidelines for acrylonitrile (refer to Appendix A).

6.3 European Union

No information about acrylonitrile guidelines in European member countries was found.

6.4 Australia/ New Zealand

The New Zealand Ministry of Environment and Ministry of Health recently proposed guidelines for selected air toxics; however, acrylonitrile was not included (New Zealand, 2000).

Table 6-1 Summary of Air Quality Guidelines for Acrylonitrile (refer to Appendix A for agency reference)

Agency	Guideline Title	Guideline Value [μg/m³] Averaging Time:			
		30-min	1-hour	24-hour	Annual
Ontario MOE	Ambient air quality criterion (AAQC):			0.6	0.12
	Maximum point of impingement (POI):	180			
US ATSDR	Acute (1 to 14 day exposure) inhalation minimum risk level (MRL):			217	
US EPA	Reference Concentration (RfC):				2
	Risk specific concentration (RsC):[1]				0.14
California EPA	Chronic reference exposure level (REL):				2
	Risk specific concentration (RsC):[1]				0.03
Louisiana DEQ	Ambient air standard (AAS):				1.47
Michigan DEQ	Initial threshold screening level (ITSL):			2	
	Initial risk screening level (IRSL):				0.01
	Secondary risk screening level (SRSL):				0.1
New Hampshire DES	24-hour ambient air limit (AAL):			15	
	Annual AAL:				2
New Jersey DEP	Risk assessment approach is used:				
	Hazard quotient (HQ):				2
	Risk specific concentration (RsC):[1]				0.14
North Carolina ENR	Acceptable ambient level (AAL):				0.15
Ohio EPA	Risk specific concentration (RsC):[1]				0.14
Oklahoma DEQ	Maximum acceptable ambient concentration (MAAC):			43	
Rhode Island DEM	Acceptable ambient level (AAL):				0.01
Texas Natural Resource Conservation Commission (TRNCC)	Effects screening level (ESL):		43		4.3
Vermont ANR	Hazardous ambient air standard (HAAS):				0.015
Washington State DOE	Acceptable source impact level (ASIL):				0.015
Wisconsin DNR	Ambient air concentration (AAC):[2]				2

[1] The RsC is not used for any specific purposes by the respective agency. It is shown here to illustrate an exposure concentration in air associated with an inhalation unit risk factor used by the agency and a 1 in 100,000 lifetime cancer risk (risk criteria used in Alberta)

[2] Proposed

NOTE: 1 μg/m³ = 0.45 ppb at 25°C and 1 atmosphere.

7.0 DISCUSSION

When establishing an ambient air guideline in the form of a concentration limit with a corresponding duration (i.e. averaging time), a number of factors may be taken into account for an air pollutant:

- nature of adverse health effects and conditions of exposure (e.g. exposure concentrations and duration) associated with these effects,
- estimated or actual degree of exposure of receptors, and in particular receptor groups that may be sensitive to the air pollutant,
- available technologies and associated economics for routinely or periodically monitoring for the pollutant in air,
- availability and suitability of approaches for screening and estimating ambient ground-level concentrations in order to compare to the guidelines for permit applications or other situations.

Ambient air guidelines in the form of a short-term (acute) and long-term (chronic) duration are discussed below for acrylonitrile. Ideally the guidelines would serve to address exposures related to humans, animals and vegetation. No direct exposure-related information was obtained for vegetation, therefore the discussion emphasizes human and animal (as surrogates for human) exposures.

7.1 Acute Exposure Conditions

There are no reported facilities in Alberta emitting acrylonitrile above reporting thresholds set for the National Pollutant Release Inventory (NPRI, 1999). As indicated previously, it may be possible that acrylonitrile is emitted in Alberta in such small amounts that the facilities are not required to report to the NPRI.

With regard to acute, short-term exposure, animal studies indicate that acrylonitrile has caused salivation, tearing of the eyes, diarrhea, gastrointestinal injury, seizures, and death. These types of responses have been observed at concentrations ranging from 58 to 1,330 mg/m^3 (26 to 600 ppm) over exposure durations ranging from 4 hrs. to 60 hrs. (on an intermittent basis). Adverse health responses have been reported in humans after acute (accidental) exposure via inhalation and/or skin contact. These responses include eye and throat irritation, central nervous system effects (e.g. headaches, nausea, vomiting, convulsions and unconsciousness), and death. Corresponding doses (exposure concentrations and durations) associated with these events are not known with a great deal of certainty because exposures were accidental and air concentrations could not be measured. With respect to non-occupational circumstances, acute exposure conditions are unlikely for the general population because of the absence of major sources.

The majority of agencies reviewed do not have an air quality guideline for acrylonitrile for acute exposure conditions. Two agencies in the US (New Hampshire DES and Oklahoma DEQ)

adopted 24-hour guidelines for acrylonitrile from occupational exposure limits (OELs). The use of OELs for the development of ambient guidelines is cautioned. There are limitations in the direct and indirect application of OELs for ambient air quality guidelines for a number of reasons:

- OELs are based on the information gathered in workplace, through experience from medical research and practice, from experimental human and animal studies, and from a combination of these sources. Often they are based upon averaged tolerated doses from actual repeated industrial exposures. In this respect, they would be considered very accurate at predicting human adverse health effects in industrial exposure situations.
- OELs are determined for a population of workers who are essentially healthy and who fall within a working age group of about 17 to 65 years. These individuals are supposedly in the prime of life, and potentially less susceptible to the effects of hazardous substances than other members of the public. Individuals vary in sensitivity or susceptibility to hazardous substances, with the elderly and infants in general being more susceptible than healthy workers.
- For most substances, a worker during a normal work schedule (8 hours per day, 5 days per week) receives 40 hours of exposure per week with daily breaks and extended weekend periods in which the body may rid itself of the accumulated substances before elevated levels are reached. For a person living continuously in an environment containing such substances; however, these recovery periods do not exist.

For these reasons, agencies using OELs have a policy of adjusting them downward with the use of safety or adjustment factors to derive guidelines for environmental (ambient) settings. The OELs are considered surrogates for benchmark values for ambient exposures only because they tend to be based upon a large body of toxicological, epidemiological, and/or clinical evidence pertaining to human exposure (albeit in the workplace). Uncertainty exists in terms of whether too much (or too little) safety is inherent in ambient air guidelines developed from OELs.

7.2 Chronic Exposure Conditions

Chronic, long-term exposures have been demonstrated to cause different cancers in a number of rat studies, including tumour formation in the brain and spinal cord, small intestine, tongue, Zymbal gland, mammary glands. Air concentrations associated with these cancers ranged from 45 to 178 mg/m^3 (20 to 80 ppm) for exposure durations of one to two years (on an intermittent basis). Long-term human exposure has occurred in occupational settings. Results of these studies are inconclusive in terms of establishing acrylonitrile's ability to cause cancer because of confounding factors (specifically, exposure to other chemicals and lifestyle factors such as smoking). Notwithstanding, data from these studies are used by US Environmental Protection Agency to represent excess incidence of respiratory cancers in humans corresponding to 33 mg/m^3 (15 ppm) as an 8-hour time weighted average concentration for an average exposure duration of 9 years. Most regulatory agencies consider acrylonitrile to be potentially carcinogenic to humans due to evidence of carcinogenicity in rat studies.

Chronic, long-term exposures have also been demonstrated to cause different adverse responses in several rat studies, including irritation of eye and skin; developmental, neurological, respiratory, and systemic effects; and death. Air concentrations associated with these responses ranged from 45 to 178 mg/m^3 (20 to 80 ppm) for exposure durations of one to two years (on an intermittent basis).

All of the agencies whose air quality guidelines were reviewed have chronic (long-term) guidelines for acrylonitrile. Almost all use a carcinogenic endpoint to derive their respective guideline – using the US EPA's inhalation unit risk of 6.8E-0.5 per $\mu g/m^3$. Several of the agencies also have a chronic guideline for non-carcinogenic effects – using the US EPA's reference concentration (2 $\mu g/m^3$ or 0.9 ppb). These agencies include California EPA, New Hampshire DES, New Jersey DEP, and Wisconsin DRN. The RfC is based on degeneration and inflammation of nasal respiratory epithelium in rats exposed through inhalation.

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APPENDIX A

REVIEW OF AIR QUALITY GUIDELINES FOR ACRYLONITRILE USED BY AGENCIES IN NORTH AMERICA AND ELSEWHERE

Agency: Ontario Ministry of the Environment (OME).
Acrylonitrile Air Quality Guideline: Annual ambient air quality criterion (AAQC) = 0.12 $\mu g/m^3$. 24-hour ambient air quality criterion (AAQC) = 0.6 $\mu g/m^3$. 30-minute Maximum point of impingement (POI) = 180 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: See above.
Basis for Development: The Ontario OME accepted the scientific rationale and risk assessment procedures employed by Environment Canada and Health Canada in EC (2000) in the development of tumourigenic concentration (TC_{05}) values for the tumour-causing effect of acrylonitrile. A tumorigenic concentration (TC_{05}) is the concentration generally in air associated with a 5% increase in incidence or mortality due to tumours. TC_{05}s divided by a suitable margin provide a benchmark against which the adequacy of exposure to ambient or indoor air can be judged, with respect to potential carcinogenicity. The application of safety factors of 5,000 and 50,000 to the TC_{05} produces risk concentrations of 1.2 and 0.12 $\mu g/m^3$, representing excess cancer risks of 1 in 100,000 and 1 in 1,000,000, respectively.
Date Guideline Developed: March 2001.
How Guideline is Used in Practice: The AAQC are used by Ontario Ministry of Environment (OME) to represent human health or environmental effect-based values not expected to cause adverse effects based on continuous exposure.
Additional Comments: The AAQC <u>are not</u> used by OME to permit stationary sources that emit acrylonitrile to the atmosphere. The 30-minute Maximum POI is derived by mathematical scaling from an ambient air quality criterion (AAQC) and is used by OME to review permit applications for stationary sources that emit acrylonitrile to the atmosphere.
Reference and Supporting Documentation: Ontario Ministry of the Environment (Ontario MOE). 2001. Ontario Air Standards for Acrylonitrile. Standards Development Branch, Ontario Ministry of the Environment. Toronto, ON. March 2001. 70 pp. EC (Environment Canada). 2000. *Priority Substances List Assessment Report: Acrylonitrile.* Canadian Environmental Protection Act, 1999.Environment Canada, Health Canada. February 1999.

Agency: US Agency for Toxic Substances and Disease Registry (ATSDR).
Acrylonitrile Air Quality Guideline: Acute inhalation minimum risk level (MRL) = 0.1 ppm (217 $\mu g/m^3$).
Averaging Time To Which Guideline Applies: 1 to 14 day exposure durations.
Basis for Development: Absence of neurological effects. In a study with humans volunteers exposed to acrylonitrile at does of 2.3 to 4.6 ppm, no symptoms attributable to effects on the nervous system were observed. The dose of 4.6 ppm was used to calculate the acute inhalation MRL of 0.1 ppm. After adjusting for intermittent exposure, the dose was divided by a safety factor of 10 for human variability to arrive at an acute inhalation MRL of 0.1 ppm.
Date Guideline Developed: December 1990.
How Guideline is Used in Practice: MRLs are intended to serve as a screening tool to help public health professionals decide where to look more closely. Acute inhalation MRLs are exposure concentrations that, based on current information, might cause adverse health effects in the people most sensitive to such substance-induced effects for 1 to 14 day exposure durations.
Additional Comments: Inhalation MRLs provide a basis for comparison with levels that people might encounter in air. If a person is exposed to acrylonitrile at an amount below the MRL, it is not expected that harmful (noncancer) health effects will occur. Because these levels are based only on information currently available, some uncertainty is always associated with them. Also, because the method for deriving MRLs does not use any information about cancer, an MRL does not imply anything about the presence, absence, or level of risk for cancer.
Reference and Supporting Documentation: Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological Profile for Acrylonitrile. ATSDR, Public Health Service, US Department of Health and Human Services. Atlanta, GA. December 1990. 141 pp.

Agency: US Environmental Protection Agency (EPA).
Acrylonitrile Air Quality Guideline: Reference concentration (RfC) = 2 $\mu g/m^3$. Risk specific concentration (RsC) corresponding to 1 in 100,000 risk = 0.14 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: Continuous exposure (daily exposure over a lifetime).
Basis for Development: The RfC was developed as follows. A two-year inhalation exposure study to rats identified a LOAEL (20 ppm) based on degeneration and inflammation of nasal respiratory epithelium in rats exposure through inhalation. A human equivalent concentration (HEC) of 1.9 mg/ m^3 was adjusted with an uncertainty factor of 1000 to derive a RfC of 2 $\mu g/m^3$ after rounding. The RsC corresponding to 1 in 100,000 risk (risk criteria used in Alberta) was derived in the following manner. Data from epidemiologic study of respiratory cancer from inhalation in the workplace were used. An inhalation unit risk of 6.8E-05 per ($\mu g/m^3$) was used with 70-kg body weight adult breathing 20 m^3/day. The inhalation unit risk was calculated from a relative risk model using data from human studies adjusted for smoking and based on a continuous lifetime equivalent of occupational exposure.
Date Guideline Developed: Last revised in 1991.
How Guideline is Used in Practice: The reference concentration (RfC) and inhalation unit risk are intended for use by US EPA staff in risk assessments, decision-making and regulatory activities. The risk specific concentration (RsC) is not used for any specific purposes by US EPA and is shown here to illustrate an exposure concentration in air associated with an inhalation unit risk factor derived by US EPA and a 1 in 100,000 lifetime cancer risk.
Additional Comments: The Integrated Risk Information System (IRIS) is prepared and maintained by the US EPA. IRIS is an electronic database containing information on human health effects that may result from exposure to various chemicals in the environment.
Reference and Supporting Documentation: US Environmental Protection Agency. Integrated Risk Information System. http://www.epa.gov/iris/ (accessed 8 August 2001).

Agency:

California Environmental Protection Agency (Cal EPA).

Acrylonitrile Air Quality Guideline:

Chronic reference exposure level (REL) = 2 µg/m³.
Risk specific concentration (RsC) corresponding to 1 in 100,000 risk = 0.03 µg/m³.

Averaging Time To Which Guideline Applies:

Continuous exposure (daily exposure over a lifetime).

Basis for Development:

The basis for the chronic REL is protection against respiratory irritation (CAPCOA, 1993). The RsC corresponding to 1 in 100,000 risk (risk criteria used in Alberta) was derived as follows. Data from an epidemiologic study of respiratory cancer from inhalation in the workplace were used. Cal EPA re-calculated an inhalation unit risk of 2.9E-04 per (µg/m³) using the same data considered by US EPA. This value was used with a 70-kg body weight adult breathing 20 m³/day. The inhalation unit risk was re-calculated by Cal EPA from a relative risk model using data from human studies adjusted for smoking and based on a continuous lifetime equivalent of occupational exposure.

Date Guideline Developed:

Chronic REL – 1992.
Inhalation unit risk – 1999.

How Guideline is Used in Practice:

Chronic RELs are for use in facility health risk assessments conducted for the AB 2588 Air Toxics "Hot Spots" Program. The risk specific concentration (RsC) is not used for any specific purposes by Cal EPA and is shown here to illustrate an exposure concentration in air associated with an inhalation unit risk factor derived by Cal EPA and a 1 in 100,000 lifetime cancer risk.

Additional Comments:

n/a

Reference and Supporting Documentation:

California Environmental Protection Agency (Cal EPA). 1999. Air Toxics Hot Spots Program Risk Assessment Guidelines Part II. Technical Support Document for Describing Available Cancer Potency Factors. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, California EPA. Oakland, CA. April 1999.

California Air Pollution Control Officers Association (CAPCOA). 1993. Air Toxics Hot Spots Program Revised 1992 Risk Assessment Guidelines. CAPCOA Toxics Committee, Cameron Park, CA. October 1993.

Agency: Louisiana Department of Environmental Quality (DEQ).
Acrylonitrile Air Quality Guideline: Ambient air standard (AAS) for toxic air pollutants = 1.47 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: Annual average.
Basis for Development: Not stated.
Date Guideline Developed: Not stated.
How Guideline is Used in Practice: AASs are used by Louisiana DEQ to review permit applications for stationary sources that emit acrylonitrile to the atmosphere.
Additional Comments: n/a
Reference and Supporting Documentation: *Louisiana Administrative Code (LAC). Title 33 Environmental Quality, Part III Air, Chapter 51. Comprehensive Toxic Air Pollutant Emission Control Program. Louisiana Department of Environmental Quality. Baton Rouge, LA.*

Agency:

Michigan Department of Environmental Quality (DEQ).

Acrylonitrile Air Quality Guideline:

Initial threshold screening level (ITSL) = 2 µg/m³ [24-hour averaging time].
Initial risk screening level (IRSL) = 0.01 µg/m³ [annual averaging time].
Secondary risk screening level (SRSL) = 0.1 µg/m³ [annual averaging time].

Averaging Time To Which Guideline Applies:

See above.

Basis for Development:

The ITSL is based on the US EPA reference concentration of 2 µg/m³. The IRSL and SRSL are based on the US EPA inhalation unit risk of 6.8E-05 per (µg/m^3) and lifetime cancer risks of one in one million (10^{-6}) and 1 in 100,000 (10^{-5}), respectively.

Date Guideline Developed:

Not stated.

How Guideline is Used in Practice:

There are two basic requirements of Michigan air toxic rules. First, each source must apply the best available control technology for toxics (T-BACT). After the application of T-BACT, the emissions of the toxic air contaminant cannot result in a maximum ambient concentration that exceeds the applicable health based screening levels (ITSL, IRSL, or SRSL). Application of an ITSL is required for any new or modified emission source or sources for which a permit to install is requested and which emits a toxic air contaminant. The IRSL applies only to the new or modified source subject to the permit application. If an applicant cannot demonstrate that the emissions of the toxic air contaminant meet the IRSL, they may choose to demonstrate compliance with the SRSL; however, in this case they must include all sources of that toxic air contaminant emitted from the plant, not just the emission unit being permitted.

Additional Comments:

The applicable air quality screening level for chemical treated as non-carcinogens by Michigan DEQ is the ITSL. There are two health based screening levels for chemical treated as carcinogens by Michigan DEQ: the IRSL – based on an increased cancer risk of one in one million (10^{-6}), and the SRSL – based on as an increased cancer risk of 1 in 100,000 (10^{-5}).

Reference and Supporting Documentation:

Michigan Administrative Code (MAC). Air Pollution Control Rules. Part 2 Air Use Approval, R 336.1201 - 336.1299. Air Quality Division, Department of Environmental Quality. Lansing, MI.

Agency: New Hampshire Department of Environmental Services (DES).
Acrylonitrile Air Quality Guideline: 24-hour ambient air limit (AAL) = 15 µg/m³. Annual ambient air limit (AAL) = 2 µg/m³.
Averaging Time To Which Guideline Applies: See above.
Basis for Development: In the case of acrylonitrile, the AALs were developed in the following manner: 24-hour Ambient Air Limit – The American Conference of Governmental Industrial Hygienists (ACGIH) 8-hour time weighted average occupational exposure limit (OEL) of 4.3 mg/m³ is divided by a safety factor (SF) of 100 and a time adjustment factor (TAF) of 2.8. Annual Ambient Air Limit – The reference concentration (RfC) limit established by the US Environmental Protection Agency is used as the annual AAL.
Date Guideline Developed: May 1998.
How Guideline is Used in Practice: AALs are used by New Hampshire DES to review permit applications for sources that emit acrylonitrile to the atmosphere. Sources are regulated through a statewide air permitting system and include any new, modified or existing stationary source, area source or device.
Additional Comments: n/a
Reference and Supporting Documentation: *New Hampshire Administrative Rule. Chapter Env-A 1400. Regulated Toxic Air Pollutants. New Hampshire Department of Environmental Services. Concord, NH.*

Agency: New Jersey Department of Environmental Protection (DEP).
Acrylonitrile Air Quality Guideline: Applicants are required to carry out a risk assessment in conjunction with applying for an air pollution control pre-construction permit. In the case of acrylonitrile, the US Environmental Protection Agency RfC (2 $\mu g/m^3$) is used as the pollutant-specific reference concentration in which to calculate a Hazard Quotient for sources that emit acrylonitrile to the atmosphere. The US EPA inhalation unit risk factor of 6.8E-05 per ($\mu g/m^3$) is used to calculate a lifetime cancer risk for sources that emit acrylonitrile to the atmosphere.
Averaging Time To Which Guideline Applies: Continuous exposure (daily exposure over a lifetime).
Basis for Development: Based on US EPA Integrated Risk Information System (IRIS) data.
Date Guideline Developed: December 1994.
How Guideline is Used in Practice: Used by New Jersey DEP to review permit applications for sources that emit acrylonitrile to the atmosphere.
Additional Comments: n/a
Reference and Supporting Documentation: *New Jersey Administrative Code (NJAC). Title 7, Chapter 27, Subchapter 8. Permits and Certificates for Minor Facilities (and Major Facilities without an Operating Permit). New Jersey Department of Environmental Protection. Trenton, NJ.* New Jersey Department of Environmental Protection. 1994. Technical Manual 1003. Guidance on Preparing a Risk Assessment for Air Contaminant Emissions. Air Quality Permitting Program, Bureau of Air Quality Evaluation, New Jersey Department of Environmental Protection. Trenton, NJ. Revised December 1994.

Agency: North Carolina Department of Environment and Natural Resources (ENR).
Acrylonitrile Air Quality Guideline: Acceptable ambient level (AAL) = 0.15 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: Annual average.
Basis for Development: Not stated. Although not stated, the AAL was likely derived by using an increased cancer risk of one in 100,000 (10^{-5}) and the US EPA's inhalation unit risk factor of 6.8E-05 per ($\mu g/m^3$) for a 70-kg body weight adult breathing 20 m^3/day.
Date Guideline Developed: 1990.
How Guideline is Used in Practice: A facility emitting acrylonitrile must limit its emissions so that the resulting modeled ambient levels at the property boundary remain below the health-based acceptable ambient level (AAL).
Additional Comments: n/a
Reference and Supporting Documentation: *North Carolina Administrative Code (NCAC). North Carolina Air Quality Rules 15A NCAC 2D.1100 – Air Pollution Control Requirements (Control of Toxic Air Pollutants). North Carolina Department of Environment and Natural Resources. Raleigh, NC.* *North Carolina Administrative Code (NCAC). North Carolina Air Quality Rules 15A NCAC 2Q.0700 – Air Quality Permit Procedures (Toxic Air Pollutant Procedures). North Carolina Department of Environment and Natural Resources. Raleigh, NC.*

Agency: Ohio Environmental Protection Agency (EPA).
Acrylonitrile Air Quality Guideline: For chemicals treated as carcinogens – like acrylonitrile – applicants are required to carry out a risk assessment in conjunction with applying for a permit to determine the probability of incidence of a health effect(s) as a consequence of the new emission using US EPA unit risk values. In the case of acrylonitrile, the US EPA inhalation unit risk factor of 6.8E-05 per ($\mu g/m^3$) is used to calculate a lifetime cancer risk. Risk specific concentration (RsC) corresponding to 1 in 100,000 risk = 0.14 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: Continuous exposure (daily exposure over a lifetime).
Basis for Development: The RsC corresponding to 1 in 100,000 risk (risk criteria used in Alberta) was derived using the US EPA inhalation unit risk of 6.8E-05 per ($\mu g/m^3$) and a 70-kg body weight adult breathing 20 m^3/day.
Date Guideline Developed: January 1994.
How Guideline is Used in Practice: The risk specific concentration (RsC) is not used for any specific purposes by Ohio EPA and is shown here to illustrate an exposure concentration in air associated with the inhalation unit risk factor used by Ohio EPA and a 1 in 100,000 lifetime cancer risk.
Additional Comments: n/a
Reference and Supporting Documentation: Ohio Environmental Protection Agency (Ohio EPA). 1994. Review of New Sources of Air Toxic Emissions. Proposed for Public Comment. Division of Air Pollution Control, Ohio EPA. Columbus, OH. January 1994. 31 pp.

Agency: Oklahoma Department of Environmental Quality (DEQ).
Acrylonitrile Air Quality Guideline: Maximum acceptable ambient concentration (MAAC) = 43 µg/m³.
Averaging Time To Which Guideline Applies: 24-hour averaging time.
Basis for Development: The American Conference of Governmental Industrial Hygienist (ACGIH) TLV – 8-hour time weighted average occupational exposure limit (OEL) of 4.3 mg/m³ – is divided by a safety factor of 100.
Date Guideline Developed: Not stated.
How Guideline is Used in Practice: MAACs are used by Oklahoma DEQ to review permit applications for sources that emit acrylonitrile to the atmosphere.
Additional Comments: n/a
Reference and Supporting Documentation: *Oklahoma Administrative Code (OAC). Title 252. Chapter 100. Air Pollution Control. 100:252-41 - Control of Emission of Hazardous and Toxic Air Contaminants. Oklahoma Department of Environmental Quality. Oklahoma City, OK.*

Agency: Rhode Island Department of Environmental Management (DEM).
Acrylonitrile Air Quality Guideline: Acceptable ambient level (AAL) = = 0.01 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: Annual average.
Basis for Development: n/a
Date Guideline Developed: Last amended in 1992.
How Guideline is Used in Practice: AALs are used by Rhode Island DEM to review permit applications for sources that emit acrylonitrile to the atmosphere.
Additional Comments: n/a
Reference and Supporting Documentation: *Rhode Island Department of Environmental Management. 1992. Air Pollution Control Regulation No. 22. Division of Air and Hazardous Materials, Rhode Island Department of Environmental Management. Providence, RI. Amended 19 November 1992.*

Agency: Texas Natural Resource Conservation Commission (TRNCC).
Acrylonitrile Air Quality Guideline: Short-term effects screening level (ESL) = 43 µg/m³. Long-term effects screening level (ESL) = 4.3 µg/m³.
Averaging Time To Which Guideline Applies: 1-hour averaging time for short-term ESL. Annual averaging time for long-term ESL.
Basis for Development: Short-term Effects Screening Level – The American Conference of Governmental Industrial Hygienist (ACGIH) TLV – 8-hour time weighted average occupational exposure limit (OEL) of 4.3 mg/m³ – is divided by a safety factor of 100. Long-term Effects Screening Level – The ACGIH TLV – 8-hour time weighted average occupational exposure limit (OEL) of 4.3 mg/m³ – is divided by a safety factor of 1000.
Date Guideline Developed: Not stated.
How Guideline is Used in Practice: ESLs are used to evaluate the potential for effects to occur as a result of exposure to concentrations of constituents in air. ESLs are based on data concerning health effects, odor nuisance potential, effects with respect to vegetation, and corrosion effects. They are not ambient air standards. If predicted or measured airborne levels of a chemical do not exceed the screening level, adverse health or welfare effects would not be expected to result. If ambient levels of constituents in air exceed the screening levels, it does not necessarily indicate a problem, but rather, triggers a more in-depth review.
Additional Comments: n/a
Reference and Supporting Documentation: Texas Natural Resource Conservation Commission (TNRCC) 2001. Toxicology & Risk Assessment (TARA) Section Effects Screening Levels. http://www.tnrcc.state.tx.us/permitting/tox/index.html (accessed 6 September 2001).

Agency: Vermont Agency of Natural Resources.
Acrylonitrile Air Quality Guideline: Hazardous ambient air standard (HAAS) = 0.015 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: Annual average.
Basis for Development: The HAAS for known or suspected carcinogens (such as acrylonitrile) is set at a level which represents an excess risk of one additional cancer case per one million exposed population (10^{-6}) assuming constant exposure at the HAAS concentration for a lifetime. Although not stated, it was derived by using US EPA's inhalation unit risk factor of 6.8E-05 per ($\mu g/m^3$) and a 70-kg body weight adult breathing 20 m^3/day.
Date Guideline Developed: Not stated.
How Guideline is Used in Practice: HAASs are used by Vermont ANR to review permit applications for stationary sources that emit acrylonitrile to the atmosphere.
Additional Comments: n/a
Reference and Supporting Documentation: Vermont Air Pollution Control Regulations. 2001. State of Vermont Agency of Natural Resources. Air Pollution Control Division. Waterbury, VT. 29 November 2001. 187 pp.

Agency: Washington State Department of Ecology (DOE).
Acrylonitrile Air Quality Guideline: Acceptable source impact level (ASIL) = 0.015 μg/m³.
Averaging Time To Which Guideline Applies: Annual average.
Basis for Development: The ASIL for acrylonitrile is a risk-based acceptable source impact level that may cause an increased cancer risk of one in one million (10^{-6}) using US EPA's inhalation unit risk factor of 6.8E-05 per ($\mu g/m^3$) and a 70-kg body weight adult breathing 20 m^3/day.
Date Guideline Developed: September 1991.
How Guideline is Used in Practice: ASILs are used by Washington State DOE to review permit applications for sources that emit acrylonitrile to the atmosphere.
Additional Comments: n/a
Reference and Supporting Documentation: *Washington Administrative Code (WAC). Chapter 173-460 WAC. Controls For New Sources Of Toxic Air Pollutants. Washington State Department of Ecology. Olympia, WA.*

Agency: Wisconsin Department of Natural Resources (DNR).
Acrylonitrile Air Quality Guideline: Ambient air concentration (AAC) = 2 µg/m³ (proposed).
Averaging Time To Which Guideline Applies: Continuous exposure (daily exposure over a lifetime).
Basis for Development: The AAC proposed for acrylonitrile is based on the US EPA reference concentration of 2 µg/m³.
Date Guideline Developed: Not stated.
How Guideline is Used in Practice: AACs are used by Wisconsin DNR to review permit applications for sources that emit acrylonitrile to the atmosphere.
Additional Comments: n/a
Reference and Supporting Documentation: *Wisconsin Administrative Code (WAC). Air Pollution Control Rules. Chapter NR 445. Control of Hazardous Pollutants. Wisconsin Department of Natural Resources. Madison WI.*

Agency: New Zealand Ministry for the Environment and New Zealand Ministry of Health.
Acrylonitrile Air Quality Guideline: Ambient air quality guidelines are proposed for selected air toxics; however, acrylonitrile is not included.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: n/a
Reference and Supporting Documentation: New Zealand Ministry for the Environment and Ministry of Health (New Zealand). 2000. Proposals for Revised and New Ambient Air Quality Guidelines. Discussion Document. Air Quality Technical Report No 16. Prepared by the Ministry for the Environment and the Ministry of Health. December 2000. 86 pp.